Exhibit 18

UNITED STATES DISTRICT COURT
EASTERN DISTRICT OF PENNSYLVANIA

IN RE: NATIONAL FOOTBALL LEAGUE PLAYERS' CONCUSSION INJURY LITIGATION	No. 2:12-md-02323-AB MDL No. 2323
Kevin Turner and Shawn Wooden, *on behalf of themselves and others similarly situated,* Plaintiffs, v. National Football League and NFL Properties, LLC, successor-in-interest to NFL Properties, Inc., Defendants.	Hon. Anita B. Brody Civil Action No. 2:14-cv-00029-AB
THIS DOCUMENT RELATES TO: ALL ACTIONS	

DECLARATION OF JOHN G. KEILP, PHD

JOHN G. KEILP, PhD, hereby declares as follows:

1. I have personal knowledge concerning the matters addressed herein, and submit this declaration in connection with Plaintiffs' motion for approval of the proposed settlement of claims in this litigation. If called as a witness, I could and would testify competently to the facts herein.

Education, Training, & Experience

2. I am a Clinical Psychologist and Neuropsychologist. I am a Research Scientist at the New York State Psychiatric Institute, and an Assistant Professor of Clinical Psychology at the Columbia University College of Physicians and Surgeons. I am head of the

Neuropsychology Laboratory in the Division of Molecular Imaging and Neuropathology at the New York State Psychiatric Institute. I have been a licensed psychologist in New York State for twenty-four years, with an independent practice in both clinical and neuropsychological assessment in addition to my research work.

3. I received my PhD in Clinical Psychology from Fordham University in 1990. As part of this training, I completed a two-year integrated internship/fellowship in Clinical Psychology at the New York Hospital/Cornell Medical Center, with my fellowship year concentrated on neuropsychological studies of schizophrenia. I thereafter completed a two-year fellowship in Neuropsychology at the Memorial Sloan-Kettering Cancer Center. I have held faculty positions at the Mount Sinai School of Medicine and Columbia University College of Physicians and Surgeons, as well as adjunct faculty positions at Fordham University, Adelphi University, and Queens College of the City University of New York.

4. I have primarily worked in Clinical Neuropsychological research throughout my career. My research experience encompasses studies of the neuropsychological features of dementia, infectious diseases affecting the central nervous system (i.e. Human Immunodeficiency Virus, Lyme disease), schizophrenia, depression, and suicidal behavior. I have 71 peer-reviewed publications in medical, psychological, and neuropsychological journals. I have been a principal investigator or investigator on 24 grants, from both the National Institutes of Health as well as private foundations. A central feature of my work has been the development of neuropsychological assessment batteries to assess specific aspects of the disorders we have studied. This has involved tailoring clinical batteries and finding appropriate measures to assess the critical deficits in these disorders. This work has provided me with intimate knowledge of the practical considerations of administration of these measures with difficult and impaired

populations, knowledge of the basic psychometric properties of these measures, and knowledge of the statistical issues involved in multi-test evaluations.

5. My earlier research work focused on characterizing neurocognitive changes in patients with dementia, and the relationship of these changes to underlying brain pathology. In one study, correction of current test scores for estimated premorbid ability level was critical to improving the association to underlying deficits in cerebral blood flow (Keilp JG, Prohovnik I. *Intellectual decline predicts the parietal perfusion deficit in Alzheimer's Disease*. Journal of Nuclear Medicine, 1995, 36(8): 1347-1354). In another, normative adjustments of test performance clarified differences in the correlates of different aspects of language performance in patients with dementia (Keilp JG, Gorlyn M, Alexander GA, Stern Y, Prohovnik I. *Cerebral blood flow patterns underlying the differential impairment in category vs. letter fluency in Alzheimer's disease*. Neuropsychologia, 1999, 37: 1251-1261).

6. My current work focuses on characterizing the neurocognitive impairments that accompany disorders—such as depression—that are thought to be primarily behavioral, and on the contributions of neurocognitive impairment to the risk for suicide and suicidal behavior. I published one of the first systematic studies of neurocognitive deficits associated with suicidal behavior (Keilp JG, Sackeim H A, Brodsky B, Oquendo M, Malone K, Sackeim H, Mann JJ. *Neuropsychological dysfunction in depressed suicide attempters*. American Journal of Psychiatry, 2001, 158(5): 735-741) and have an international reputation for this work. More recently, I have contributed to the development of the neuropsychological assessment battery used in the Study to Assess Risk and Resilience in Servicemembers (STARRS), a project funded by the United State Army to evaluate possible neurocognitive risk factors for suicidal behavior in new recruits (Ursano RJ, Stein MB, Heeringa S, Kessler RC, Colpe LJ, Schoenbaum M,

Cersovsky S, Cox K, Aliaga PA, Benedek, DM, Borja, S., Brown GG, Campbell-Sills L, Dempsey CL, Frank R, Fullerton CS, Gebler N, Gifford RK, Gilman SE, Holloway MG, Hurwitz PE, Jain S, Kao TC, Koenen KC, Lewandowski-Romps L, Mash HH, McCarroll JE, McLaughlin KA, Naifeh JA, Nock MK, Raman R, Rose S, Rosellini AJ, Sampson NA, Santiago P, Scanlon M, Smoller J, Thomas ML, Vegella PL, Wassel CL, Zaslavsky AM, Mann J, Oquendo M, Stanley B, Posner K, Keilp J. *The Army Study to Assess Risk and Resilience in Servicemembers (Army STARRS)*. Psychiatry, 2014, 77(2): 107-19). This project, to date, has gathered data on over 51,000 individuals within the first days of their basic training in the army; analyses of these data are currently under way.

7. Additional information on my background and credentials is available on my curriculum vitae, a copy of which is attached to this declaration.

8. The statements and opinions expressed by me in this declaration are mine alone. They do not reflect those of Columbia University, the College of Physicians and Surgeons, or the New York State Psychiatric Institute, nor any of the faculty, staff, or administration of those institutions. All opinions set forth herein I hold to a reasonable degree of scientific certainty.

The Brain, Neuropsychological Functioning, and Methods of Assessment

9. In the last fifty years, we have advanced substantially in our understanding of the close association between the brain and behavior.

10. There are various disciplines that concern themselves with this association, including neurosurgery, neurology, psychiatry, and psychology. As our knowledge of brain/behavior relationships has grown, even disciplines such as economics have begun to incorporate findings from brain sciences to better understand how the information processing

capabilities of the brain may systematically distort information that individuals use to make critical economic decisions.

11. Brain injury can lead to permanent alterations in an individual's ability to think and function on a day-to-day basis. There are also many diseases that affect central nervous system function and have an impact on both the brain and behavior. Progressive, degenerative conditions cause an increasing deterioration of cognitive function over time and can lead to permanent disability. Normal processes such as aging also affect brain function and behavior in systematic ways, and all assessments of functional disability are undertaken in the context of multiple influences on cognitive outcomes.

12. Neuropsychology broadly encompasses any attempt to relate behavior to brain function. The field concerns itself with accurately measuring both behavior and brain function, and our attempts to relate the two. The field of Clinical Neuropsychology places its emphasis on quantifying cognition and behavior, and drawing inferences about underlying pathological processes based on known patterns of cognitive impairment. Clinical Neuropsychology plays a critical role in the characterization of brain dysfunction by evaluating cognitive performance and behavior in a standard, objective, and quantitative manner.

13. For the purposes of our assessments, we typically divide neurocognition into various domains of function, with the understanding that different types of underlying brain pathology can lead to deficits or impairments in distinct domains. These domains of functioning often correspond to the activity in or integrity of specific areas of the brain, or in specific functional circuitry of the brain. For example, Alzheimer's disease in its early stages has its most pronounced effects on memory, and abnormalities of cerebral blood flow and metabolism are typically concentrated in temporal and parietal regions associated with memory functioning.

Other aspects of cognitive functioning will be affected in the earliest stages of Alzheimer's disease, but often to a lesser degree than memory. Because this disease is progressive, however, in its more advanced stages, Alzheimer's disease will have a profound effect on virtually all aspects of neurocognition.

14. Clinical neuropsychological assessments use psychometric tests to clarify the extent of cognitive dysfunction across functional domains. These tests include standard questions and procedures whose results are then compared to normative data, or preexisting data for the individual from before the onset of disease or injury, if available. The objective data generated from these procedures are then used to increase the precision of clinical judgments. Tests are validated by comparing performance to underlying pathology, typically in larger scale research studies. In legal settings, clinical neuropsychological assessment allows us to establish clear, standardized criteria for characterizing level of impairment relative to healthy peers who are demographically similar.

15. Clinical Neuropsychological assessments also commonly incorporate systematic procedures to evaluate the quality of effort made by examinees, and to detect attempts to mislead those making evaluative judgments via deliberately poor performance. In compensation settings, these "effort tests" become important for establishing the validity of the data collected.

Neurocognitive Disorder Injury Definitions & Baseline Assessment Program

16. Since the summer of 2013, I have consulted with Plaintiffs' Co-Lead Class Counsel concerning various scientific matters underlying the claims at issue in the instant litigation, including issues implicated in connection with any settlement of such claims. Among the matters I was asked to assist with was the construction of clinically reasonable and accepted

ways to measure the extent of impairments in retired players using psychometric testing, and objective criteria for defining the compensable impairment levels in retired players. In that regard, together and in collaboration with other medical professionals retained by Plaintiffs, I assisted with the development of the Injury Definitions for Level 1 and Level 2 neurocognitive impairment (and later, Level 1.5 neurocognitive impairment) that form the basis of the Qualifying Diagnoses in the Settlement Agreement. I further assisted in the development of the neuropsychological test battery that was ultimately incorporated into and became the framework for the Baseline Assessment Program (BAP). I am thus intimately familiar with the test battery and the related thresholds for the neurocognitive disorders incorporated in the Injury Definitions.

17. The framework for characterizing impairment was derived from the Neurocognitive Disorders section of the most recent version, the 5th revision, of the Diagnostic and Statistical Manual for Mental Disorders ("DSM-5"). This manual characterizes neurocognitive impairment with respect to its nature and severity in a manner that is informed by knowledge about particular disease states and injuries, but not exclusive to any particular disease or injury. As such, it can be used to broadly characterize the level of dysfunction in key neurocognitive domains without respect to specific etiology.

18. The DSM-5 divides neurocognition into six broad domains corresponding to common aspects of everyday cognitive function. The domains are (1) Complex Attention and Processing Speed, (2) Learning and Memory, (3) Executive Function, (4) Language, (5) Visual-Spatial and Visuomotor Function, and (6) Social Cognition.

19. In everyday terms, these domains of function address specific questions about a given patient's cognitive functioning as follows: (1) Complex Attention and Processing Speed: Is the patient alert and focused, and can they finish tasks in a reasonable amount of time? (2)

Learning and Memory: Can the patient remember things, especially new things they have just been exposed to? (3) Executive Function: Can the patient figure things out, keep things organized, and get them done in an orderly way? (4) Language: Can the patient find the right words when they speak, and understand what people are telling them? (5) Visual-Spatial and Visuomotor Function: Can the patient understand spatial relationships, figure out how to put a puzzle together, or perform rapid, coordinated motor tasks? (6) Social Cognition: Can the patient recognize and respond to social cues, or interact appropriately with other people?

20. With the exception of Social Cognition, these domains of functioning were incorporated into the Injury Definitions for neurocognitive impairments in the Settlement Agreement (at Exhibit 1), and related standardized neuropsychological testing protocol annexed in Exhibit 2 to the Settlement Agreement. Social Cognition was not included given that objective measures of social cognition are less well developed, particularly with respect to the influence of poor effort. Though a Visual-Spatial domain was included in the Injury Definitions and related test battery, motor and visuomotor functioning were deemphasized given the high rate of orthopedic injury and rheumatologic disease in former players, which would distort inferences regarding central nervous system injury that might be made from players' performance on motor tests.

21. In addition to describing domains of functioning, the DSM-5 divides neurocognitive disorders into two levels of severity. These are referred to as "Major" and "Mild" Neurocognitive Disorders, and exist on a spectrum of functional and cognitive impairment. These classifications are further described by a general level of neurocognitive impairment that can be characterized by neuropsychological tests, such that the results of neuropsychological testing can be translated to a severity level in DSM-5 terms. However, the

DSM-5 itself does not provide specific cutoffs for specific tests; rather, it specifies a general level of impairment that must be translated by the clinician for the specific tests administered.

22. Major Neurocognitive Disorders encompass a level of impairment that falls 2.0 or more standard deviations below normative expectations for a given patient. These normative expectations will differ based on a variety of factors, including the patient's age, sex, education level, and premorbid ability level. This level of cognitive impairment, when it extends beyond a single domain of function, corresponds to that commonly associated with the diagnosis of dementia (McKhann GM, Knopman DS, Chertkow H, Hyman BT, Jack CR Jr, Kawas CH, Klunk WE, Koroshetz WJ, Manly JJ, Mayeux R, Mohs RC, Morris JC, Rossor MN, Scheltens P, Carrillo MC, Thies B, Weintraub S, Phelps CH. *The diagnosis of dementia due to Alzheimer's disease: recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease*. Alzheimers Dement, 2011, 7(3): 263-9).

23. Mild Neurocognitive Disorders encompass a level of impairment that falls 1-2 standard deviations below normative expectations for a given patient. The spectrum of patient outcomes, functional impact, and progression of impairment in this range is highly variable and often dependent on the underlying etiology of the impairment. Though there is a real decline in cognitive function, many patients remain able to function at a high level personally and professionally notwithstanding detected impairments. For others, the functional impact is manifest and discernable. Though many patients with mild cognitive impairment (approximately 40%) progress to Major Neurocognitive Impairment over the course of their life, the majority do not (Mitchell, AJ & Shiri-Feshki, M. *Rate of progression of mild cognitive impairment to dementia--meta-analysis of 41 robust inception cohort studies*. 2009, Acta Psychiatrica

Scandinavica, 2009, 119(4), 252-265). The level of impairment in prior studies of mild cognitive impairment that raises the risk for progression to later dementia falls at approximately 1.5 standard deviations below population norms—the midpoint of the Mild Neurocognitive Disorder range of severity specified by the DSM-5 (Devanand D, Lee J, Luchsinger J, Manly J, Marder K, Mayeux R, Scarmeas N, Schupf N, Stern Y. *Lessons from epidemiologic research about risk factors, modifiers, and progression of late onset Alzheimer's Disease in New York City at Columbia University Medical Center*. Journal of Alzhiemer's Disease, 2013, 33 Suppl 1:S447-55.)

24. Levels of impairment in the Injury Definitions for the Settlement Agreement were extended from the principles and framework set forth in DSM-5 Neurocognitive Disorders. In doing so, efforts were made to empirically tie neuropsychometric testing results to various levels of cognitive decline specified by DSM-5, *e.g.*:

> **Level 1.0 Neurocognitive Impairment** is characterized descriptively as "a moderate cognitive decline from a previous level of performance", and is reflected by test performance in two or more cognitive domains at least 1.5 standard deviations below population norms. This level of impairment falls roughly at the 7th percentile of the normal population (based on a translation of standard deviation units into percentiles).
>
> **Level 1.5 Neurocognitive Impairment** is characterized descriptively as "a moderate to severe cognitive decline from a previous level of performance", and is reflected by test performance in two or more cognitive domains at least 1.7-1.8 standard deviations below population norms. This level of impairment falls roughly at the 5th percentile of the normal population (based on a translation of standard deviation units into percentiles), and clinically corresponds to early symptoms of dementia

> **Level 2.0 Neurocognitive Impairment** is characterized descriptively as "a severe cognitive decline from a previous level of performance", and is reflected by test performance that falls 2.0 or more standard deviations below population norms. This level of impairment falls roughly at the 2nd percentile of the normal population (based on a translation of standard deviation units into percentiles), and clinically corresponds to moderate dementia.

See Settlement Agreement, Exhibit 1 at 1-4 (Injury Definitions for Level 1, 1.5, and 2); Exhibit 2 at 5 (Neuropsychological Test Battery, Section 4).

25. Beyond the presence of cognitive impairments detected through neuropsychological testing, the Injury Definitions define other items necessary for each of the Qualifying Diagnoses. In particular, the Injury Definitions require that (a) the retired player or other knowledgeable informant is concerned about cognitive decline in the retired player; (b) the retired player exhibits functional impairment in connection with aspects of his life (personal care, home life, hobbies, and community affairs); and (c) the detected cognitive impairments do not appear exclusively as a result of some acute condition or event (*e.g.*, delirium, acute substance abuse, medication side effects). *See* Settlement Agreement, Exhibit 1 at 1-4 (Injury Definitions for Level 1, 1.5, and 2). These requirements were modeled on the framework for Mild and Major Neurocognitive Disorders from the DSM-5.

26. Under the Settlement, the presence of neurocognitive impairments can be assessed within the framework of the Settlement's Baseline Assessment Program (BAP), or outside the BAP by qualified physicians using the BAP test battery or other neuropsychological tests to evaluate the relevant cognitive domains and existence of deficits.

27. The neuropsychological test battery developed for the BAP (Settlement Agreement, Exhibit 2) designates specific neuropsychological measures to assess functioning in each of the five domains derived from the DSM-5 and relevant to the Injury Definitions. Multiple measures are used in each domain in order to insure adequate coverage of each domain, and a reliable determination of level of function within each domain. This is a common practice in Clinical Neuropsychology.

28. Measures selected for the BAP test battery are among the most widely used in Clinical Neuropsychology, and are widely available to clinicians throughout the country (Rabin LA, Barr WB, Burton LA. *Assessment practices of clinical neuropsychologists in the United States and Canada: a survey of INS, NAN, and APA Division 40 members*. Archives of Clinical Neuropsychology, 2005, 20(1): 33-65). Measures selected are those with the most extensive normative data, so that accurate determinations of level of impairment can be made uniformly for former players being tested through the BAP.

29. The BAP assessment battery includes measures designed to assess five domains of functioning: (1) Complex Attention and Processing Speed, (2) Learning and Memory, (3) Executive Functioning, (4) Language, and (5) Spatial-Perceptual Functioning. Standard, well-known tests with extensive use in clinical settings and extensive normative data were selected to characterize functioning in each of these domains.

30. Impairment for each neurocognitive injury level was defined empirically based on a specific number of tests within a domain falling below a "cutpoint" indicative of impairment. These cutpoints were selected based on the aggregate likelihood that 7% (for Level 1.0), 5% (for Level 1.5), or 2% (for Level 2.0) of the normal population would have scores falling below that level. (Brooks, BL, Iverson, GL, Holdnack JA. *Understanding and Using Multivariate Base*

Rates with the WAIS-IV/WMS-IV, pp 75-102 in J Holdnack, L Drozdick , L Weiss , G Iverson WAIS-IV, WMS-IV, and ACS, 2014, New York: Elsevier).

31. Classification at each level was also based on scoring below designated cutpoints in two domains. At least one of these domains was to reflect an impairment in Complex Attention and Processing Speed, Learning and Memory, or Executive Functioning, as these were thought to be the most likely to result from the experience of former players. A combination of impairments in Language and Spatial-Perceptual Functioning without impairment in other domains was thought to be unlikely, given that these are typically associated with injuries on opposite sides of the brain (MD Lezak, Neuropsychological Assessment, 4th Edition, 2004, New York: Oxford).

32. Many of the tests selected also have data available regarding the effects of inconsistent effort, or deliberate attempts at deception. Such testing is commonly employed to assess the quality of the effort that the patient provided and to evaluate the validity of the results obtained with a given test battery. Given the compensation setting in which the BAP test battery is being deployed, it is fair and reasonable to do so here.

33. The determination of cognitive impairment in any individual depends on a variety of factors. It is well known, for example, that premorbid ability has a profound effect on the expression of deficits following brain injury or disease. Thus, in attempting to discern the extent of any cognitive impairment in a particular patient, it is standard practice in all neuropsychological assessments to estimate premorbid ability. (Brooks, BL, Iverson, GL, Holdnack JA. *Predicting Premorbid Ability for WAIS-IV, WMS-IV and WASI-II*, pp 217-278 in J Holdnack, L Drozdick , L Weiss , G Iverson WAIS-IV, WMS-IV, and ACS, 2014, New York: Elsevier; Iverson, G. & Brooks, B., *Improving Accuracy for Identifying Cognitive Impairment*,

pp 923-950 in M Schoenberg & J Scott ed. The Little Black Book of Neuropsychology: A Syndrome-Based Approach. 2011, New York: Springer). Accordingly, the BAP test battery not only includes tests of specific neurocognitive functions, but also measures to estimate basic ability level that may have preceded any injuries (premorbid ability level). Empirical data on these effects is available and was used in the construction of the BAP assessments. A key consideration in the construction of cutpoints for impairment was the existence of empirical data on the frequency of low demographically-adjusted scores in different premorbid ability groups. Data tables on the effects of premorbid ability on performance across multiple tests within critical domains in the BAP were obtained from Dr. Grant Iverson, an expert on the effects of premorbid ability on the prevalence of low test scores across a test battery (even when scores are demographically adjusted) (Brooks, BL, Iverson, GL, Holdnack JA. *Understanding and Using Multivariate Base Rates with the WAIS-IV/WMS-IV*, pp 75-102 in J Holdnack, L Drozdick , L Weiss , G Iverson WAIS-IV, WMS-IV, and ACS, 2014, New York: Elsevier). Dr. Iverson, an expert retained by Plaintiffs' Co-Lead Counsel, further consulted in the development of the test battery, construction of cutpoints, and Injury Definitions.

34. The BAP includes supplemental measures of behavioral dysfunction, in the form of both a self-report psychopathology scale (Minnesota Multiphasic Personality Inventory, 2nd Edition, Revised Format or "MMPI 2-RF") as well as a structured psychiatric interview (Mini International Neuropsychiatric Interview or "MINI"). Although behavioral disorders are not compensated, they are not penalized. The inclusion of the MMPI 2-RF and MINI, which rely on subjective reports from the particular patient is, in my opinion, a sound decision. Depressive and behavioral disorders can impact cognitive function. Early identification and treatment of such disorders can improve the management of cognitive conditions and overall functioning

prospectively in retired players. For retired players qualifying for Level 1 supplemental benefits under the BAP, obtaining such information can guide further testing and care. In the case of all retired players, these neuropsychiatric measures provide valuable information concerning conditions that are often treatable.

35. The BAP also incorporates consideration of functional impairment outside the realm of cognition alone. To qualify at a specific level of impairment, former players are required to exhibit functional impairment consistent with neurocognitive impairment. The standards used in the construction of these functional impairment criteria are derived from the Clinical Dementia Rating scale ("CDR"; Morris JC. *The clinical dementia rating (CDR): current version and scoring rules*. Neurology, 1993, 43: 2412–2414) a well validated and commonly-used scale for assessing the progression of dementia symptoms (Williams MM, Roe CM, Morris JC. *Stability of the Clinical Dementia Rating, 1979-2007*. Archives of Neurology, 2009, 66(6): 773-7). These functional impairment criteria correspond to the functional impairments typically seen in "Questionable" dementia (CDR level .5 and Level 1.0 Injury Definition in the Settlement scheme), "Mild" dementia (CDR level 1.0 and Level 1.5 Injury Definition in the Settlement scheme), and "Moderate" dementia (CDR level 2.0 and Level 2.0 Injury Definition in the Settlement scheme).

36. I have administered the test battery and applied the thresholds specified under the Settlement Agreement with retired players, and reviewed data from other neuropsychologists who have administered this battery as well. Practically, it performs as intended. Patients with definable neurocognitive impairments are able to tolerate its length and complete it in good order. Moreover, thresholds for determining level of impairment correlate well with the clinical reality and presentation of these patients. Score thresholds correspond with my clinical

experience examining the performance of patients with moderate neurocognitive impairment, early dementia, and moderate to severe dementia. A patient who will satisfy the Level 2 Injury Definition by testing is likely to be diagnosed with moderate dementia. Similarly, a patient who will satisfy the Level 1.5 Injury Definition by testing is most likely to be in the early stages of dementia. In my opinion, practicing neuropsychologists will readily and reliably be able to implement the test battery under the BAP with retired players, using the skill, training, and experience they would employ in their day-to-day clinical practice.

Response to Critiques

37. The BAP and neurocognitive injury definitions have been criticized for not specifically testing living retired players for Chronic Traumatic Encephalopathy (CTE), an emerging neuropathological finding reported from the post-mortem examination of the brains of deceased people, often athletes and veterans. The BAP and neurocognitive injury definitions, however, were not designed to assess any specific neuropathology, but to define a level of impairment consistent with any number of causes. The BAP test battery specifically relies on neurocognitive assessment because neurocognitive impairment is a relevant outcome from the type of injuries at issue in this case, and can be assessed in an objective, quantifiable manner with safeguards against attempts to falsify impairment.

38. Suggestions that CTE can be "diagnosed" based on behavioral symptoms that have a high base rate in former football players (and, in many instances, the general population) are misguided. The science and clinical standards concerning CTE are in their infancy, and have yet to reach acceptance. Although technological improvements in assessing brain pathology may aid the process of characterizing this disorder, substantial additional research is needed to

characterize it. Longitudinal studies to chart its course, prospective and developmental studies to determine the onset of any pathological changes in the brain, and population studies to determine the prevalence of CTE in athletes from other sports as well as non-athletes with repetitive or even single incident head trauma will be needed. In addition, if CTE pathology is progressive and neurocognitive deterioration symptomatically accompanies such progression, the BAP and Injury Definitions are likely to detect these changes with its progression.

39. The BAP test battery and neurocognitive injury definitions have been criticized for not including behavioral disorders in determining compensation. With regard to the assessments themselves, measures of behavioral impairment are included in a self-report measure (MMPI2-RF) and a structured clinical interview (MINI) that are part of the BAP. Both measures include questions on irritability, lowered inhibitions, and suicidal thinking. Though the neurocognitive injury definitions do not recognize behavioral disorders as independent qualifying diagnoses, such symptoms and conditions commonly accompany neurocognitive impairments in the progressive dementias. Even if they do not, they can become the focus of follow-up care and additional treatment, which is of benefit to the retired player.

40. The BAP test battery has been criticized as being too long and difficult for former players who are most impaired and likely to qualify for the highest levels of compensation. This criticism is not well founded. Batteries of this type have been administered to individuals who meet criteria for dementia. Trained and experienced neuropsychologists commonly confront patients with this level of impairment. I have personally conducted research in patients with Alzheimer's using these and analogous test measures (see Keilp JG, Alexander GE, Stern Y, Prohovnik I. *Inferior parietal perfusion, lateralization, and neuropsychological dysfunction in Alzheimer's disease*. Brain and Cognition, 1996, 32(3):365-83). Though people with advanced

impairments may fail at many of the tasks, they nonetheless produce scoreable performance. Finally, many of the tests themselves contain rules and criteria to distinguish dementia from invalid cognitive performance.

41. The BAP test battery has been criticized as having inconsistent impairment criteria. However, impairment criteria were derived from empirical tables establishing base rates in the normal population for low performance (below the 7th, 5th, or 2nd percentile, respectively, for each of the three impairment levels) on multiple tests within a single domain, stratified by premorbid ability level. (Brooks, BL, Iverson, GL, Holdnack JA. *Understanding and Using Multivariate Base Rates with the WAIS-IV/WMS-IV*, pp 75-102 in J Holdnack, L Drozdick , L Weiss , G Iverson WAIS-IV, WMS-IV, and ACS, 2014, New York: Elsevier)

42. The BAP test battery has been criticized as relying exclusively on a reading test to establish premorbid ability level. However, in addition to the administration of the Advanced Clinical Solutions (ACS) Word Reading Test, premorbid ability of former players will also be estimated using demographic formulas available in the ACS scoring package. These estimates—one based on a simple demographic formula and one based on a more detailed demographic formula—will provide alternative means of assessing premorbid ability. The procedure producing the highest premorbid estimate will be used to determine the appropriate criteria for assessing level of impairment.

43. The BAP has been criticized as having excessive effort testing. However, due to the level of compensation, it is important to incorporate safeguards against deliberate attempts to manipulate the outcome of the assessment procedures. The scope of this settlement encompasses many former players and will continue for many years. It was necessary to incorporate sufficient measures to assess the quality of effort to insure the prospective integrity of this program.

44. The inclusion of effort testing in the BAP has been criticized because, at some point, effort testing is susceptible to the effects of real impairment. However, the BAP incorporates the Slick criteria for assessing effort, which takes into account the overall pattern of performance and its concordance with known patterns of impairment. (Slick DJ, Sherman EM, Iverson GL. *Diagnostic criteria for malingered neurocognitive dysfunction: proposed standards for clinical practice and research*. Clinical Neuropsychology. 1999, 13(4): 545-61). These criteria incorporate clinical observation by the neuropsychologist. Neuropsychological assessment in the BAP will only be administered by diplomate level, board-certified neuropsychologists, those with the most senior level of clinical credentialing. These individuals will be qualified to judge if effort testing has been failed due to the severity of their level of impairment. This may then be corroborated by an independent neurological exam.

45. The BAP test battery has been criticized for the possibility that individuals with the same raw test scores might not be eligible for the same compensation. However, it is a standard feature of any neuropsychological assessment to only judge raw scores in the context of demographic factors and estimates of premorbid ability. This approach has been validated in studies correlating levels of neuropathology to estimated declines in functioning. An individual with a higher level of education and a higher level of premorbid ability who is currently functioning at a below average level will in all likelihood have a higher level of brain pathology than an individual functioning at the same level who, premorbidly, functioned at a below average level. To fail to adjust for demographic or premorbid ability levels would penalize those with better than average ability, and provide an advantage for those who have lower premorbid ability and experience only a slight decline in function. The goal of the stratification of ability levels is to make the determinations of impairment as fair as possible.

Conclusions

A. The BAP test battery and the related Injury Definitions are a fair and objective means of determining the level of neurocognitive impairment based on standard procedures in Clinical Neuropsychology.

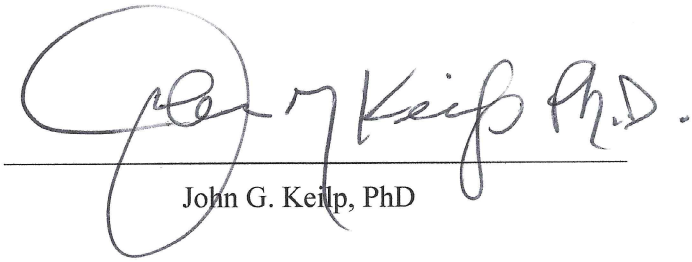
B. The BAP test battery and the related Injury Definitions are informed by, but not restricted to, any specific pathogenic etiology.

C. The BAP test battery and injury definitions are focused on ascertaining and compensating serious functional and neurocognitive impairments affecting retired players during their lives. Though behavioral disorders do not independently inform and determine the level of neurocognitive impairment under the injury definitions, behavioral disorders and impairments are likely to accompany the evaluated and serious neurocognitive impairments that the program compensates—if not in the short term, then certainly as underlying disease processes progress.

D. For those who do not meet criteria for the qualifying diagnoses, the BAP test battery allows for early detection of neurocognitive and behavioral disturbances that can become the focus of ongoing treatment. In those players whose condition progresses to more severe neurocognitive injury and a qualifying diagnosis, they and their families/representatives are able to apply for compensation throughout the life of the program.

I declare under penalty of perjury that the foregoing is true and correct.

Dated: November 12, 2014
New York, New York



John G. Keilp, PhD

Attachment

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Date of Preparation: October 29, 2014

Personal Data:

Name:	John George Keilp
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5/2001 – present	**Department of Psychiatry, Columbia University** Assistant Professor of Clinical Psychology in Psychiatry	New York, NY
11/1999 – present	**Department of Molecular Imaging and Neuropathology (formerly Brain Imaging/Neuroscience)** New York State Psychiatric Institute Research Scientist IV	New York, NY
11/1993 – 11/1999	**Department of Brain Imaging/Neuroscience** New York State Psychiatric Institute Research Scientist II	New York, NY
9/1993 – 5/2001	**Department of Psychiatry, Columbia University** Associate Research Scientist	New York, NY
7/1992 – 7/1993	**Department of Psychiatry, Mount Sinai School of Medicine** Assistant Professor of Psychiatry	New York, NY
4/1990 – 6/1992	**Department of Psychiatry, Mount Sinai School of Medicine** Instructor of Psychiatry	New York, NY
4/1990 – 7/1993	**Mount Sinai Services, Elmhurst Hospital Center** Senior Psychologist	Queens, NY
6/1989 – 3/1990	**Laboratory of Clinical Psychophysiology** New York Hospital/Cornell Medical Center Research Associate	New York, NY
7/1986 – 6/1987	**Laboratory of Psychopharmacology** New York Hospital/Cornell Medical Center Staff Associate	New York, NY

Education:

9/1982 – 12/1989	**Fordham University, Graduate School of Arts and Sciences** Ph.D. in Clinical Psychology, January 1990 Ph.D. Thesis Title: "Cerebral Ventricular Enlargement in Schizophrenia: Relationships to Neuropsychological Functioning, Symptomatology, and Disruptions in Speech Prosody" Sponsors: Marvin Reznikoff, Ph.D., John Sweeney, Ph.D., John Sidtis, Ph.D.	Bronx, NY
9/1981 – 9/1982	**Fordham University, Graduate School of Arts and Sciences** M.A. in Clinical Psychology, January 1983	Bronx, NY
9/1976 – 5/1978	**Cornell University** Graduate Study, Human Development and Family Studies	Ithaca, NY
9/1972 – 5/1976	**Rutgers University** B.A. in Psychology	New Brunswick, NJ

Training:

7/1987 – 5/1989	**Department of Neurology, Memorial Sloan-Kettering Cancer Center** Fellow in Neuropsychology	New York, NY
7/1984 – 6/1986	**Payne Whitney Clinic, New York Hospital/Cornell Medical Center** Intern/Fellow in Clinical Psychology	New York, NY

Between Graduate Programs:

6/1978 – 9/1981	**Various Employment** City of Syracuse, New York (Public Information Specialist) Family and Children's Services of Syracuse (Counselor) New York State Division for Youth (Aide) City of Ithaca, New York (Swimming Instructor/Lifeguard)

Licensure:

8/1990 – present	Licensed Psychologist, New York State (#010203-1)

Honors:

1999	Sallie Foundation Young Investigator Award
1989	Sigma Xi
1983	Phi Kappa Phi
1983	University Teaching Fellowship (Fordham University)
1981, 1982	Graduate Assistantships (Fordham University)
1977, 1978	Graduate Assistantships (Cornell University)
1976	Phi Beta Kappa (Rutgers University)

Grant Support:

Present Support:

7/2013 – 6/2018
Conte Center: Neurobiological and Developmental Antecedents to Suicidal Behavior
National Institute of Mental Health (NIMH)
1P50MH090964-01A1
Role: Co-Investigator (J.J. Mann, Principal Investigator)
Direct Support Year 01: $1,348,516

7/2013 – 7/2015
Ketamine vs. Midazolam: Testing Rapid Relief of Suicide Risk in Bipolar Disorder
Brain-Behavior Research Foundation (BBRF/NARSAD)
BBRF 12-3845
Role: Co-Investigator (M. Grunebaum, Principal Investigator)
Direct Support Year 01: $50,000

7/2002 – 1/2015
Familial Pathways to Early-Onset Suicide Attempts
National Institute of Mental Health (NIMH)
5R01MH056390-15
Role: Co-Investigator (J.J. Mann, Principal Investigator)
Direct Support Year 05: $237,828

7/2012 – 4/2017
Ketamine vs. Midazolam: Testing Rapid Relief of Suicide Risk in Depression
National Institute of Mental Health (NIMH)
5R01MH096784-03
Role: Co-Investigator (M. Grunebaum, Principal Investigator)
Direct Support Year 03: $390,459

12/2008 – 11/2014
Future Suicide Attempt: Psychobiological Features
National Institute of Mental Health (NIMH)
5R01MH048514-20
Role: Investigator (M. Oquendo, Principal Investigator)
Direct Support Year 05: $237,600

Past Support:

7/2008 – 4/2014
Treating Suicidal Behavior and Self-Mutilation in BPD
National Institute of Mental Health (NIMH)
5R01MH061017-10
Role: Investigator (B. Stanley, Principal Investigator)
Total Direct Support: $2,676,574

7/2007 – 12/2013
Aggressive Behavior in Depression and Suicide: Biochemical, Behavioral, and Cognitive Aberrations in the Stress Response
National Alliance for Research on Schizophrenia and Depression (NARSAD)
Role: Mentor (M. Gorlyn, Principal Investigator)
Total Direct Support: $60,000

2/2006 – 1/2011 Paroxetine/Buproprion in Suicide Attempters/Ideators in Major Depression
National Institute of Mental Health (NIMH)
5K23MH076049-05
Role: Mentor/Consultant (M. Grunebaum, Principal Investigator/K Award)
Total Direct Support: $818,653

7/2000 – 6/2010 Conte Center for the Neuroscience of Mental Disorders (CCNMD):
The Neurobiology of Suicidal Behavior
National Institute of Mental Health (NIMH)
5P50MH062185-10
Role: Investigator (J.J. Mann, Principal Investigator)
Total Direct Support: $7,460,460 (last five years)

12/2006 – 11/2008 Neuropsychological Predictors of Antidepressant Treatment Response in Suicide Attempters
American Foundation for Suicide Prevention (AFSP)
Role: Mentor (M. Gorlyn, Principal Investigator)
Total Direct Support; $70,000

7/1996 – 6/2008 Neuropsychological Characteristics of Suicide
National Alliance for Research on Schizophrenia and Depression (NARSAD)
Role: Principal Investigator
Total Direct Support: $60,000

7/1998 – 6/2008 Localization of Selective Attention Deficits in High Lethality Suicide Attempters
National Alliance for Research on Schizophrenia and Depression (NARSAD)
Role: Principal Investigator
Total Direct Support: $60,000

7/1996 – 6/2008 Pharmacotherapy of High-Risk Bipolar Disorder
National Institute of Mental Health (NIMH)
5R01MH059710-05
Role: Investigator (M. Oquendo, Principal Investigator)
Total Direct Support: $2,900,000

7/1996 – 6/2008 Neuropsychological Characteristics of Suicide
National Alliance for Research on Schizophrenia and Depression (NARSAD)
Role: Principal Investigator
Total Direct Support: $60,000

7/2005 – 7/2007 Naturalistic Study of Duration of Untreated Psychosis and Neuroleptic Response in Schizophrenia
National Alliance for Research on Schizophrenia and Depression (NARSAD)
Role: Co-Mentor (with A. Dwork/B. Mancevski, Principal Investigator)
Total Direct Support: $60,000

4/2002 – 5/2007 Neuropsychological Dysfunction in Suicidal Behavior
National Institute of Mental Health (NIMH)
5R01MH062155-04
Role: Principal Investigator
Total Direct Support: $400,000

4/1999 – 3/2004 PET and MRI Imaging of Persistent Lyme Encephalopathy
National Institute of Neurological Disorders and Stroke (NINDS)
5R01NS038636-04
Role: Investigator (B. Fallon, Principal Investigator)
Total Direct Support: $4,998,216

4/1999 – 3/2004 Prefrontal D_1 and $5HT_{1A}$ Receptors in Schizophrenia
National Institute of Mental Health (NIMH)
5R01MH059144-03
Role: Investigator (A. Abi-Dargham, Principal Investigator)
Total Direct Support: $1,527,936

7/2000 – 6/2003 Assessment of Prefrontal Cortical Dysfunction in Suicidal Behavior
American Foundation for Suicide Prevention (AFSP)
Role: Principal Investigator
Total Direct Support: $60,000

12/1998 – 2/2003 AD-Like Pathology in Elderly Schizophrenia
National Institute of Mental Health (NIMH)
5R01MH060877-08
Role: Investigator (A. Dwork, Principal Investigator)
Total Direct Support: $1,630,948

7/1998 – 6/2000 Center for the Study of Suicidal Behavior
National Institute of Mental Health (NIMH)
5P30MH046745-11
Role: Investigator (J.J. Mann, Principal Investigator)
Total Direct Support: $3,098,500

1/1998 – 4/1998 Pilot Study: Neuropsychological Assessment during Treatment with VPA-985
Unrestricted Grant from Wyeth-Ayerst Research
Role: Investigator (J.J. Mann, Principal Investigator)
Total Direct Support: $150,000

12/1996 – 9/1998 Pilot Study: Neuropsychological Assessment during Treatment with Dexfenfluramine
Unrestricted Grant from Interneuron Pharmaceuticals
Role: Investigator (J.J. Mann, Principal Investigator)
Total Direct Support: $106,536

12/1996 – 12/1997 Impulsivity Assessment in the Study of Suicide
New York State Psychiatric Center MHCRC Seed Grant
Role: Principal Investigator
Total Direct Support: $5,000

9/1992 – 8/1994 Neurobiology and the Etiology of Schizophrenia
Scottish Rite Schizophrenia Research Program (at Mt. Sinai Medical Center)
Role: Investigator (B. Cornblatt, Principal Investigator)
Total Direct Support: $60,000

Pending Support:

10/2014 – 9/2016 Neurocognitive Markers of Vulnerability to Suicidal Behavior Across the Life-Cycle
Linked Standard Research Grant, American Foundation for Suicide Prevention (AFSP)
Role: Principal Investigator (3 Site, Multi-Site Study)
Total Direct Support: $75,000 (each site)

Teaching Experience:

Teaching:

1995 – 2014 Annual Presentations: Works in Progress Seminar Series, Division of Molecular Imaging and Neurpathology (formerly Neuroscience), New York State Psychiatric Institute.

2012 – 2013 Guest Lecturer: Dialectical Behavior Therapy Course (Residents and Psychology Interns; B. Brodsky, Instructor); Neuropsychological Characteristics of Borderline Personality Disorder.

9/1983 – 5/1984 Teaching Fellow: Introductory Psychology (3) and Personality Psychology (1) Courses, Fordham University.

Invited Addresses:

6/2014 Grand Rounds: Department of Psychiatry, Duke University Medical Center, Durham, North Carolina, and Central Regional Hospital, Butner, North Carolina. Title: “How research is changing our understanding of suicidal behavior.”

5/2014 Grand Rounds: Department of Psychiatry, Columbia University College of Physicians and Surgeons, New York, New York. Title: “How research is changing our understanding of suicidal behavior.”

4/2014 Invited Speaker: Lyme Disease Association Annual Meeting, Providence, Rhode Island. Title: “Understanding suicidal behavior risk in Lyme Disease: Perspectives from studies of suicidal behavior in depression.”

4/2014 Invited Speaker: Colloquium Series, Department of Clinical Psychology, Teacher’s College, Columbia University. Title: “Neurocognition in suicidal behavior: Where does it fit?”

5/2013	Invited Speaker: Macedonian Psychiatric Association 5th Annual Meeting, Ohrid, Macedonia. Title: "Evolving perspectives on the clinical correlates of suicidal behavior."
4/2012	Invited Speaker: German Borreliosis Society Annual Meeting, Schweinfurt, Germany. Title: "Differentiating neurocognitive deficits in post-treatment Lyme disease syndrome from psychiatric comorbidities."
10/2008	Invited Speaker: Conference *Lyme and Other Tick-Borne Diseases: Solutions through Cutting-Edge Science* sponsored by the Lyme Disease Association, San Francisco, California. Title: "Neuropsychological profile of Lyme disease vs. depression."
5/2007	Invited Speaker: Conference *Neuropsychological and Neuropsychiatric Impact of Autoimmune Disorders* sponsored by the New York Academy of Sciences and New York Neuropsychology Group, New York, New York. Title: "Cognitive problems in Lyme disease and depression."
1/2006	Grand Rounds: Department of Psychiatry, Wake Forest University School of Medicine, Winston-Salem, North Carolina. Title: "The perplexing relationship of impulsiveness to suicidal behavior."
10/1999	Invited Speaker: Colloquium Series, Department of Psychology, Queens College, City University of New York, Queens, New York. Title: "Neuropsychological assessment and suicidal behavior."
12/1994	Grand Rounds: Department of Psychiatry, Elmhurst Hospital Center, Queens, New York. Title: "Imaging studies with the continuous performance test."
1/1993	Grand Rounds: Department of Psychiatry, Elmhurst Hospital Center, Queens, New York. Title: "Differentiating neurological and psychiatric symptomatology in AIDS-related dementia."
3/1990	Grand Rounds: Department of Psychiatry, Elmhurst Hospital Center, Queens, New York. Title: "Vocal prosody in schizophrenia: Relationships to affective flattening, cerebral ventricular enlargement, and neuropsychological performance."
12/1988	Grand Rounds: Department of Psychiatry, Elmhurst Hospital Center, Queens, New York. Title: "Neuropsychological assessment of AIDS-related dementia."

Thesis/Dissertation Mentorship:

2013	Diane Scheiner, Ph.D. Dissertation: "The Incremental Contribution of Posttraumatic Stress Disorder to Verbal Learning and Memory Performance Profiles in Major Depression" (granted by Fordham University).

2009	Efrat Schori, Ph.D. Dissertation: "Association of Self-Report Measures of Psychosis-Proneness and the COMT Gene Val158/Met Polymorphism" (granted by Yeshiva University).
2006	Lee Damsky, Ph.D. Dissertation: "Attachment, Control of Attention, and Self-Regulation: What is the Nature of the Relationship?" (granted by the New School for Social Research).
2007	Marina Schickman, Ph.D. Dissertation: "Age, Gender, General Intelligence, and Educational Influences on Working Memory" (granted by the City University of New York).
2006	Lee Damsky, M.A. Thesis: "Differentiating Depressed Patients with, and without, Borderline and non-Borderline Personality Disorders using Attachment Self-Report" (granted by the New School for Social Research)
2004	Sofia Marsano, Ph.D. Dissertation: "Differentiating the Neuropsychological Testing Patterns of Borderline Personality Disorder and Major Depressive Disorder" (granted by Fordham University).
2003	Marcela Bonafina, Ph.D. Dissertation: "Time Estimation in Schizophrenia: Relationship to Clinical and Neuropsychological Functioning" (granted by the City University of New York).
2003	Gwinne Wyatt (Porter), Ph.D. Dissertation: "Executive Functioning in Depression and Suicide" (granted by Fordham University).
2001	Marianne Gorlyn, Ph.D. Dissertation: "Performance Test Correlates of Impulsivity and its Component Factors: (granted by Fordham University)
1998	Felice Tager, Ph.D. Dissertation: Neuropsychological Deficits in Children with Lyme Disease" (granted by Yeshiva University).
1998	Nilima Ramaswamy, M.A. Thesis: "Association of BDRS Factors with rCBF in Patients with Alzheimer's Disease" (granted by New York University).
1997	Rice Fuller, M.A. Thesis: "Regional Cerebral Blood Flow Correlates of Memory Decline in the Normal Elderly" (granted by Fordham University).
1997	Marianne Gorlyn, M.A. Thesis: "Frontal Lobe Perfusion and Neuropsychological Dysfunction in Alzheimer's Disease" (granted by Fordham University).
1995	Annagret Brown, Ph.D. Dissertation: "Adjustment to Diagnosis of Alzheimer's Disease in Spouses of AD Patients" (granted by Adelphi University).

Publications:
Original Peer-Reviewed Papers:

1. Brent, D.A., Wyatt, G., Melhem, N.M., Oquendo, M., Burke, A., Birmaher, B., Stanley, B., Biernesser, C., Keilp, J. Kolko, D., Ellis, S., Porta, G., Zelazny, J., Iyengar, S., Mann, J.J. Familial pathways to early onset suicide attempt: A 5.6 year prospective study. JAMA Psychiatry, In press. (no PMID yet)

2. *Keilp, J.G., Wyatt, G., Oquendo, M.A., Harkavy-Friedman, J., Mann, J.J. Intact alternation performance in high lethality suicide attempters. Psychiatry Research, 2014, 219(1), 129-136. PMID: 24878299

3. Gill, K.E., Cressman, V., Poe, S.L., Steinfeld, S., Ben-David, S., Keilp J.G., Moore, H., Turkstra, L., Corcoran, C.M. Social inference in individuals at clinical high risk for psychosis. Early Intervention in Psychiatry, In press. (No PMID yet)

4. Ursano, R.J., Stein, M.B., Heeringa, S., Kessler, R.C., Colpe, L.J., Schoenbaum, M., Cersovsky, S., Cox, K., Aliaga, P.A., Benedek, D.M., Borja, S., Brown, G.G., Campbell-Sills, L., Dempsey, C.L., Frank, R., Fullerton, C.S., Gebler, N., Gifford, R.K., Gilman, S.E., Holloway, M.G., Hurwitz, P.E., Jain, S., Kao, T.C., Koenen, K.C., Lewandowski-Romps, L., Mash, H.H., McCarroll, J.E., McLaughlin, K.A., Naifeh, J.A., Nock, M.K., Raman, R., Rose, S., Rosellini, A.J., Sampson, N.A., Santiago, P., Scanlon, M., Smoller, J., Thomas, M.L., Vegella, P.L., Wassel, C.L., Zaslavsky, A.M., Mann, J., Oquendo, M., Stanley, B., Posner, K., Keilp, J. The Army Study to Assess Risk and Resilience in Servicemembers (Army STARRS). Psychiatry, 2014, 77(2), 107-19. PMID 24865195

5. Bruder, G.E., Alvarenga, J.E., Alschuler, D., Abraham, K., Keilp, J.G., Hellerstein, D.J., Stewart, J.W., McGrath, P.J. Neurocognitive predictors of antidepressant clinical response. Journal of Affective Disorders, 2014, 166, 108-114. PMID: 25012418

6. *Keilp, J.G., Beers, S.R., Burke, A.K., Melhem, N.M., Oquendo, M.A., Brent, D.A., Mann, J.J. Neuropsychological deficits in past suicide attempters with varying levels of depression severity. Psychological Medicine, 2014, 44(14), 2965-74. PMID: 25066266

7. Scheiner, D.L., Keilp, J.G., Mindt, M.R., Burke, A.K., Oquendo, M.A., Mann, J.J. Verbal learning deficits in posttraumatic stress disorder and depression. Journal of Traumatic Stress, 2014, 27(3), 291-298. PMID: 24850268

8. Sublette, M.E., Galfalvy, H.C., Hibbeln, J.R., Keilp, J.G., Oquendo, M.A., Mann, J.J. Polyunsaturated fatty acid assocations with dopaminergic indices in major depressive disorder. International Journal of Neuropsychopharmacology, 2014, 17(3), 383-391. PMID: 24300434.

9. Fallon, B.A., Petkova, E., Keilp, J.G., Britton, C.B. Ongoing discussion about the U.S. clinical Lyme trials [Letter]. American Journal of Medicine, 2014, 127(2), e7. PMID: 24462018.

10. Chandra, A.M., Keilp, J.G., Fallon, B.A. Correlates of perceived health-related quality of life in post-treatment Lyme encephalopathy. Psychosomatics, 2013, 54(6), 552-559. PMID: 23845316.

11. Gorlyn, M., Keilp, J.G., Oquendo, M.A., Burke, A.K., Mann, J.J. Iowa Gambling Task performance in currently depressed suicide attempters. Psychiatry Research, 2013, 207(3), 150-157. PMID: 23489594.

12. Grunebaum, M.F., Keilp, J.G., Ellis, S.P., Sudol, K., Bauer, N., Burke, A.K., Oquendo, M.A., Mann, J.J. SSRI versus bupropion effects on symptom clusters in suicidal major depressive disorder: post hoc analysis of a randomized clinical trial. Journal of Clinical Psychiatry, 2013, 74(9), 872-879. PMID: 24107760.

13. *Keilp, J.G., Gorlyn, M., Russell, M., Harkavy-Friedman, J., Oquendo, M.A., Mann, J.J. Neuropsychological function and suicidal behavior: Attention control, memory, and executive dysfunction in suicide attempt. Psychological Medicine, 2013, 43(3): 539-551. PMID: 22781400

14. Kikuchi, T., Miller, J.M., Schneck, N., Oquendo, M.A., Mann, J.J., Parsey, R.V., Keilp J.G. Neural responses to incongruency in a blocked-trial Stroop fMRI task in major depressive disorder. Journal of Affective Disorders, 2012, 143(1-3): 241-247. PMID: 22995943

15. Fallon, B.A., Petkova, E., Keilp, J.G., Britton, C.B. A Reappraisal of the U.S. Clinical Trials of Post-Treatment Lyme Disease Syndrome. Open Neurology, 2012, 6: 79-87. PMID: 23091568

16. *Keilp, J.G., Grunebaum, M., Gorlyn, M., LeBlanc, S., Burke, A.K., Galfalvy, H., Oquendo, M.A., Mann, J.J. Suicidal ideation and the subjective aspects of depression. Journal of Affective Disorders, 2012, 140(1): 75-81. PMID: 22406338

17. Culang-Reinlieb, M., Sneed, J.R., Keilp, J.G., Roose, S.P. Change in cognitive functioning in depressed older adults following treatment with sertraline or nortriptyline. International Journal of Geriatric Psychiatry, 2012, 27(8): 777-784. PMID 21919060

18. Fertuck, E.A., Keilp, J., Song, I., Morris, M.C., Wilson , S.T., Brodsky, B.S., Stanley, B. Higher Executive control and visual memory performance predict treatment completion in borderline personality disorder. Psychotherapy and Psychosomatics, 2012, 81(1), 38-43. PMID: 22116411

19. Milak, M.S., Keilp, J., Parsey, R.V., Oquendo, M.A., Malone, K.M., Mann, J.J. Regional brain metabolic correlates of self-reported depression severity contrasted with clinician ratings. Journal Affective Disorders, 2010, 126(1-2), 113-124. PMID: 20381874

20. Sneed, J.R., Culang, M., Keilp, J.G., Rutherford, B.R., Devanand, D.P., Roose, S.P. Antidepressant Medication and Executive Dysfunction: A Deleterious Interaction in Late-Life Depression. American Journal of Geriatric Psychiatry, 2010, 18(2), 128-135. PMID: 20104069

21. *Keilp, J.G., Oquendo, M.A., Stanley, B.H., Burke, A.K., Cooper, T.B., Malone, K.M., Mann, J.J. Future suicide attempt and responses to serotonergic challenge. Neuropsychopharmacology, 2010, 35(5), 1063-1072. PMID: 18354392

22. Culang, M., Sneed, J.R., Keilp, J.G., Devanand, D.P., Roose, S.P. Change in cognitive functioning following acute antidepressant treatment in late-life depression. American Journal of Geriatric Psychiatry, 2009, 17(10), 881-8. PMID: 19916207

23. Fallon, B.A., Likpkin, R.B., Corbera, K.M., Yu, S., Nobler, M.S., Keilp, J., Petkova, E., Lisanby, S.H., Moeller, J.R., Slavov, I., Van Heertum, R., Mensch, B.D., Sackeim, H.A. Regional cerebral blood flow and metabolic rate in persistent Lyme encephalopathy. Archives of General Psychiatry, 2009, 66(5), 554-563. PMID: 19414715

24. Gorlyn, M., Keilp, J.G., Grunebaum, M.F., Taylor, B.P, Oquendo, M.A., Bruder, G.E., Stewart, J.W., Zalsman, G., Mann, J.J. Neuropsychological characteristics as predictors of SSRI treatment response in depressed subjects. Journal of Neural Transmission, 2008, 115(8), 1213-1219. PMID: 18629432

25. *Keilp, J.G., Gorlyn, M., Oquendo, M.A., Burke, A. K., Mann, J.J. Attention deficit in depressed suicide attempters. Psychiatry Research, 2008, 159(1-2), 7-17. PMID: 18329724

26. Sneed, J.R., Keilp, J.G., Brickman, A.M., Roose, S.P. The specificity of neuropsychological impairment in predicting antidepressant non-response in the very old depressed. International Journal of Geriatric Psychiatry, 2008, 23(3), 319-323. PMID: 17726720

27. Fallon, B.A., Keilp, J.G., Corbera, K., Petkova, E., Britton, C., Dwyer, E., Slavov, I., Cheng, J., Dobkin, J., Sackeim, H.A. A randomized, placebo-controlled trial of repeated IV antibiotic therapy for Lyme encephalopathy. Neurology, 2008, 70(13), 992-1003. PMID: 17928580

28. Sneed, J.R., Roose, S.P., Keilp, J.G., Krishnan, K.R.R., Alexopoulos, G.S., Sackeim, H.A. Response inhibition predicts poor antidepressant treatment response in the very old depressed. American Journal of Geriatric Psychiatry, 2007, 15(7), 553-563. PMID: 17586780

29. Xu, H., Kellendonk, C.B., Simpson, E., Keilp, J.G., Bruder, G.E., Polan, H.J., Kandel, E.R., Gilliam, T.C. DRD2 C957T polymorphism interacts with the COMT Val158Met polymorphism in human working memory ability. Schizophrenia Research, 2007, 90(1-3), 104-107. PMID: 17113268

30. *Keilp, J.G., Klain, H.M., Brodsky, B., Oquendo, M.A., Gorlyn, M., Stanley, B., Mann, J.J. Early visual information processing deficits in depression with and without borderline personality disorder. Psychiatry Research, 2007, 149, 139-145. PMID: 17097149

31. Sackeim, H.A., Prudic, J., Fuller, R., Keilp, J., Lavori, P.W., Olfson, M. The cognitive effects of electroconvulsive therapy in community settings. Neuropsychopharmacology 2007, 32, 244-254. PMID: 16936712

32. Mancevski, B., Keilp, J., Videnova,V., Rodzevski, K., Harkavy-Friedman, J., Ortakov, V., Rosoklija, G., Dwork, A.J. Lifelong course of positive and negative symptoms in chronically institutionalized patients with schizophrenia. Psychopathology, 2007, 40, 83-92. PMID: 17215594

33. *Keilp, J.G., Gorlyn, M., Oquendo, M.A., Brodsky, B., Ellis, S.P., Stanley, B., Mann, J.J. Aggressiveness, not impulsiveness or hostility, distinguishes suicide attempters with major depression. Psychological Medicine, 2006, 36, 1779-1788. PMID: 16959059

34. Harkavy-Friedman, J.M., Keilp, J.G., Grunebaum, M.F., Sher, L., Printz, D., Burke, A. K., Mann, J.J., Oquendo, M.A. Are BP I and BP II suicide attempters distinct neuropsychologically? Journal of Affective Disorders, 2006, 94, 255-259. PMID: 16750271

35. Gorlyn, M., Keilp, J.G., Oquendo, M.A., Burke, A.K., Sackeim, H.A., Mann, J.J. The WAIS-III in major depression: Absence of VIQ/PIQ differences. Journal of Clinical and Experimental Neuropsychology, 2006, 28(7), 1145-1157. PMID: 16840241

36. Fertuk, E.A., Marsano-Jozefowicz, N.S., Tryon, W.W., Stanley, B.A., Oquendo, M.A., Mann, J.J., Keilp, J.G. The impact of borderline personality disorder and anxiety on neuropsychological performance in major depression. Journal of Personality Disorders, 2006, 20(1), 55-70. PMID: 16563079

37. *Keilp, J.G., Corberra, K., Slavov, I., Taylor, M.J., Sackeim, H.A., Fallon, B.A. WAIS-III and WMS-III performance in chronic Lyme disease. Journal of the International Neuropsychological Society, 2006, 12, 119-129. PMID: 16433951

38. Rosoklija, G., Keilp, J.G., Toomayan, G., Mancevski, B., Haroutunian, V., Liu, D., Malespina, D., Hays, A.P., Sadiq, S., Latov, N., Dwork, A.J. Altered subicular MAP2 immunoreactivity in schizophrenia. Prilozi, 2005, 26(2), 13-34. PMID: 16400226

39. Bruder, G.E., Keilp, J.G., Xu, H., Shikhman, M., Schori, E., Gorman, J.M., Gilliam, T.C. Catechol-O-Methyltransferase (COMT) genotypes: Associations to specific cognitive operations in working memory. Biological Psychiatry, 2005, 58(11), 901-907. PMID: 16043133

40. *Keilp, J.G., Sackeim, H., Mann, J.J. Correlates of trait impulsiveness in performance measures and neuropsychological tests. Psychiatry Research, 2005, 135, 191-201. PMID: 15996748

41. Grunebaum, M.F., Keilp, J.G., Li, S., Ellis, S.P., Burke, A.K., Oquendo, M.A., Mann, J.J. Symptom components of standard depression scales and past suicidal behavior. Journal of Affective Disorders, 2005, 87, 73-82. PMID: 15923041

42. Gorlyn, M., Keilp, J.G., Tryon, W.W., Mann, J.J. Performance test correlates of component factors of impulsiveness. Personality and Individual Differences, 2005, 38, 1549-1559. (No PMID/Indexed in PsycInfo)

43. Milak, M.S., Parsey, R.V., Keilp, J., Oquendo, M.A., Malone, K.M., Mann, J.J. Neuroanatomical correlates of psychopathological components of major depressive disorder. Archives of General Psychiatry, 2005, 62, 397-408. PMID: 15809407

44. Boldrini, M., DelPace, L., Placidi, G.P.A., Keilp, J., Ellis, S.P., Signori, S., Placidi, G.F., Cappa, S.F. Selective cognitive deficits in obsessive-compulsive disorder compared to panic disorder with agoraphobia. Acta Psychiatrica Scandinavica, 2004, 109, 1-9. PMID: 15667435

45. Kegeles, L.S., Malone, K.M., Slifstein, M., Ellis, S.P., Xanthopoulos, E., Keilp, J., Campbell, C., Oquendo, M.A., Van Heertum, R.L., Mann, J.J. Responses of cortical metabolic deficits to serotonergic challenge in familial mood disorders. American Journal of Psychiatry, 2003, 160(1): 76-82. PMID: 12505804

46. Oquendo, M.A., Placidi, G.P.A., Malone, K.M., Campbell, C., Keilp, J., Brodsky, B., Kegeles, L.S., Cooper, T.B., Parsey, R.V., Van Heertum, R.L., Mann, J.J. Positron emission tomography of regional brain metabolic responses to serotonergic challenge and lethality of suicide attempts in major depression. Archives of General Psychiatry, 2003, 60(1): 14-22. PMID: 12511168

47. Fallon, B.A., Keilp, J.G., Prohovnik, I., Van Heertum, R., Mann, J.J., Liebowitz, M. Regional cerebral blood flow in chronic Lyme disease. Journal of Neuropsychiatry and Clinical Neurosciences, 2003, 15(3): 326-332. PMID: 12928508

48. Abi-Dargham, A., Mawlawi, O., Lombardo, I., Gil, R., Martinez, D., Huang, Y., Hwang, D-R., Keilp, J., Kochan, L., Van Heertum, R., Gorman, J.M., Laruelle, M. Prefrontal dopamine D1 receptors and working memory in schizophrenia. Journal of Neuroscience, 2002, 22(9), 3708-3719. PMID: 11978847

49. Tager, F.A., Fallon, B.A., Keilp, J.G., Rissenberg, M., Jones, C.R., Liebowitz, M.R. A controlled study of cognitive deficits in children with chronic Lyme disease. Journal of Neuropsychiatry and Clinical Neurosciences, 2001, 13(4): 500-507. PMID: 11748319

50. *Keilp, J.G., Sackeim, H. A., Brodsky, B., Oquendo, M., Malone, K., Sackeim, H., Mann, J.J. Neuropsychological dysfunction in depressed suicide attempters. American Journal of Psychiatry, 2001,158(5): 735-741. PMID: 11329395

51. Sackeim, H.A., Keilp, J.G., Rush, A.J, George, M.S., Marangell, L.B., Dormer, J.S., Burt, T., Lisanby, S.H., Husain, M., Collum, M., Oliver, N., Zboyan, H. The effects of vagus nerve stimulation on cognitive performance in patients with treatment-resistant depression. Neuropsychiatry, Neuropsychology and Behavioral Neurology, 2001, 14(1): 53-62. PMID: 11234909

52. Rosoklija, G., Toomayan, G., Ellis, S., Keilp, J., Mann, J.J., Latov, N., Hays, A.P., Dwork, A.J. Structural abnormalities of subicular dendrites in subjects with schizophrenia and mood disorders. Archives of General Psychiatry, 2000, 57: 349-356. PMID: 10768696

53. Fallon, B., Tager, F., Fein, L., Liegner, K., Keilp, J., Weiss, N., Liebowitz, M. Repeated antibiotic treatment in chronic Lyme disease. Journal of Spirochetal and Tick-borne Diseases, 1999, 6: 94-102. (No PMID)

54. *Keilp, J.G., Gorlyn, M., Alexander, G.A., Stern, Y., Prohovnik, I. Cerebral blood flow patterns underlying the differential impairment in category vs. letter fluency in Alzheimer's disease. Neuropsychologia, 1999, 37: 1251-1261. PMID: 10530725

55. Ortakov, V., Mancevski, B., Keilp, J., Oppenheim, S., Dwork, A.J. Application of cognitive scales to medical records of schizophrenia inpatients. Schizophrenia Research, 1999, 35:131-140. PMID: 9988850

56. Dwork, A.J., Susser, E.S., Keilp, J.G., Waniek, C., Liu, D., Kaufman, M., Zemishlany, Z., Prohovnik, I. Senile degeneration and cognitive impairment in chronic schizophrenia. American Journal of Psychiatry, 1998, 155(11):1536-1543. PMID: 9812114

57. *Keilp, J.G., Herrera, J., Lee, H.K., Stritzke, P., Cornblatt, B.A. The continuous performance test, identical pairs version (CPT-IP): III. Brain functioning during performance of numbers and shapes subtasks. Psychiatry Research: Neuroimaging, 1997, 74: 35-45. PMID: 10710161

58. Intrator, J., Hare, R., Stritzke, P., Brichtswein, K., Dorfman, D., Harpur, T., Bernstein, D., Handelsman, L., Schaeffer, C., Keilp, J.G., Rosen, J., Machac, J. A brain imaging (SPECT) study of semantic and affective processing in psychopaths. Biological Psychiatry, 1997, 42: 96-103. PMID: 9209726

59. *Keilp, J.G., Alexander, G., Stern, Y., Prohovnik, I. Inferior parietal perfusion, lateralization, and neuropsychological dysfunction in Alzheimer's disease. Brain and Cognition, 1996, 32, 365-383. PMID: 8975677

60. *Keilp, J.G., Waniek, C., Goldman, R., Alexander, G.,Wu, A., Gibbon, M., Zemishlany, Z., Susser, E., Prohovnik, I. Reliability of post-mortem chart review diagnoses of schizophrenia and dementia. Schizophrenia Research. 1995, 17(2), 221-228. PMID: 8562497

61. *Keilp, J.G. & Prohovnik, I. Intellectual decline predicts the parietal perfusion deficit in Alzheimer's Disease. Journal of Nuclear Medicine, 1995, 36(8), 1347-1354. PMID: 7629576

62. Mann, J.J., McBride, P.A., Malone, K.M., DeMeo, M., Keilp, J.G. Blunted serotonergic responsivity in depressed inpatients. Neuropsychopharmacology, 1995, 13, 53-64. PMID: 8526971

63. McBride, P.A., Brown, R.P., DeMeo, M., Keilp, J., Mieczkowski, T., Mann, J.J. The relationship of platelet 5-HT_2 receptor indices to major depressive disorder, personality traits, and suicidal behavior. Biological Psychiatry, 1994, 35, 295-308. PMID: 8011798

64. Cornblatt, B.A., Keilp, J.G. Impaired attention, genetics, and the pathophysiology of schizophrenia. Schizophrenia Bulletin, 1994, 20(1), 31-46. PMID: 8197420

65. Brew, B.J., Bhalla, R.B., Paul, M., Sidtis, J.J., Keilp, J., Sadler, A.E., Gallardo, H., McArthur, J.C., Schwartz, M.K., Price, R.W. Cerebrospinal fluid beta 2-microglobulin in patients with AIDS dementia complex: An expanded series including response to zidovudine treatment. AIDS, 1992, 6(5), 461-465. PMID: 1616651

66. Sweeney, J.A., Haas, G.L., Keilp, J.G., Long, M. An evaluation of the stability of neuropsychological functioning after acute episodes of schizophrenia: One year follow-up study. Psychiatry Research, 1991, 38, 63-76. PMID: 1682967

67. Sweeney, J.A., Brew, B.J., Keilp, J.G., Sidtis, J.J., Price, R.W. Pursuit eye movement dysfunction in HIV-1 sero-positive individuals. Journal of Psychiatry & Neuroscience, 1991, 16(5), 247-252. PMID: 1797099

68. Sweeney, J.A., Keilp, J.G., Haas, G.L., Hill, J., Weiden, P.J. Relationships between medication treatments and neuropsychological test performance in schizophrenia. Psychiatry Research, 1991, 37, 297-308. PMID: 1679950

69. Heyes, M.P., Brew, B.J., Martin, A., Price, R.W., Salazar, A.M., Sidtis, J.J., Yergey, J.A., Mouradian, M.M., Sadler, A.E., Keilp, J.G., Rubinow, D., Markey, S.P. Quinolinic acid in cerebrospinal fluid and serum in HIV-1 infection: Relationship to clinical and neurological status. Annals of Neurology, 1991, 29(2), 202-209. PMID: 1826418

70. Mann, J.J., Aarons, S., Wilner, P., Keilp, J.G., Brown, R.B., Frances, A., Perlstein, S., Kocsis, J., Sweeney, J.A. A controlled study of the antidepressant efficacy and side effects of (-)-deprenyl: A selective monoamine oxidase inhibitor. Archives of General Psychiatry, 1989, 46(1): 45-50. PMID: 2491941

71. *Keilp, J.G., Sweeney, J.A., Jacobsen, P., Solomon, C., St. Louis, L., Deck, M., Frances, A., Mann, J.J. Cognitive impairment in schizophrenia: Specific relations to ventricle size and negative symptomatology. Biological Psychiatry, 1988, 24: 47-55. PMID: 3370277

Abstracts:

1. *Keilp, J.G., Gorlyn, M., Burke, A.K., Oquendo, M.A., Mann, J.J. Clarifying the role of neurocognitive impairments in the risk for suicidal behavior. Paper presented at the biannual meeting of the European Society for the Study of Suicidal Behavior, Tallinn, Estonia, August 2014.

2. Gorlyn, M., Keilp, J.G., Grunebaum, M.F., Ellis, S.E., Burke, A.K., Oquendo, M.A., Mann, J.J. Treatment-Related Changes in Suicidal Ideation are Associated with Changes in Subjective Aspects of Depression . Paper presented at the biannual meeting of the European Society for the Study of Suicidal Behavior, Tallinn, Estonia, August 2014.

3. Gorlyn, M., Keilp, J.G., Burke, A.K., Oquendo, M.A., Mann, J.J., Grunebaum, M.F. No superiority in cognitive improvement with bupropion XL vs. paroxetine CR treatment in suicidal MDD patients. Poster presented at the annual meeting of the Society of Biological Psychiatry, New York, May 2014.

4. Pantazatos, S.P., Miller, J.M., Strupp-Levitsky, M., Kikuchi, T., Oquendo, M.A., Milak, M.S., Ogden, T., Keilp, J.G., Parsey, R.V., Mann, J.J. Serotonergic function and cognitive control: Midbrain serotonin transporter binding predicts cortical conflict-related activity. Poster presented at the annual meeting of the Society of Biological Psychiatry, New York, May 2014.

5. *Keilp, J.G., Beers, S.R., Burke, A.K., Melhem, N.M., Oquendo, M.A., Brent, D.A., Mann, J.J. Neuropsychological deficits in past suicide attempters with varying levels of depression severity. Paper presented at the 42nd annual meeting of the International Neuropsychological Society, Seattle, February 2014.

6. Gorlyn, M., Keilp, J.G., Burke, A.K., Oquendo, M.A., Mann, J.J., Grunebaum, M.F. Subjective but not objective cognitive benefit of bupropion in MDD. Poster presented at the 42nd annual meeting of the International Neuropsychological Society, Seattle, February 2014.

7. Gorlyn, M., Keilp, J.G., Burke, A.K., Oquendo, M.A., Mann, J.J., Grunebaum, M.F. Treatment-related improvement in neuropsychological functioning in suicidal depressed patients: Paroxetine vs. bupropion. Poster presented at the annual meeting of the American College of Neuropsychopharmacology, Hollywood, FL, December 2013.

8. *Keilp, J.G., Beers, S.R., Burke, A.K., Melhem, N.M., Oquendo, M.A., Brent, D.A., Mann, J.J. Neuropsychological deficits in past suicide attempters with varying levels of depression: a replication and extension. Paper presented at the annual meeting of the International Academy of Suicide Research, Montreal, June 2013.

9. Gorlyn, M., Keilp, J.G., Oquendo, M.A., Mann, J.J. Semantic fluency deficit distinguishes high-lethality suicide attempters. Paper presented at the annual meeting of the International Academy of Suicide Research, Montreal, June 2013.

10. Grunebaum, M.F., Keilp, J.G., Ellis, S.P., Sudol, K., Bauer, N., Burke, A.K., Oquendo, M.A., Mann, J.J. SSRI vs bupropion effects on symptom clusters in suicidal depression: post-hoc analysis of a randomized clinical trial. Poster presented at the annual meeting of the International Academy of Suicide Research, Montreal, June 2013.

11. Grunebaum, M.F., Keilp, J.G., Ellis, S.P., Mann, J.J., Oquendo, M.A. Assessing efficacy when the outcome measure is suicidal ideation and behavior: focus on high risk populations. Paper presented at the 53d annual meeting of the New Clinical Drug Evaluation Unit (NCDEU), Phoenix, AZ, May, 2013.

12. *Keilp, J.G., Kikuchi, T., Miller, J.M., Oquendo, M.A., Parsey, R.V., Mann, J.J. Altered functional activity during Stroop task performance in depressed suicide attempters. Poster presented at the annual meeting of the Society of Biological Psychiatry, San Francisco, May 2013.

13. Gorlyn, M., Keilp, J.G., Grunebaum, M.F., Ellis, S., Burke, A.K., Oquendo, M.A., Mann, J.J. Treatment-related changes in suicidal ideation are associated with changes in subjective aspects of depression. Poster presented at the annual meeting of the Society of Biological Psychiatry, San Francisco, May 2013.

14. Bruder, G.E., Keilp, J., Alvarenga, J.E., Alschuler, D.M., Abraham, K.,Hellerstein, D.J., Stewart, J.W., McGrath, P.J. Psychomotor Slowing as a Differential Predictor of Clinical Response to Antidepressants, Poster presented at the annual meeting of the Society of Biological Psychiatry, San Francisco, May 2013.

15. *Keilp, J.G, Szanto, K. (Moderators) Fatal Decisions: Behavioral and neural accounts of aberrant decision processes in suicidal behavior across the lifespan. Symposium presented at the biannual meeting of the European Symposium on Suicide and Suicidal Behavior, Tel Aviv, Israel, September 2012.

16. Gorlyn, M., Keilp, J.G., Burke, A.K., Mann, J.J., Grunebaum, M.F. Changes in cognition and suicidal ideation during psychopharmacological treatment in depressed suicidal patients. Paper presented at the biannual meeting of the European Symposium on Suicide and Suicidal Behavior, Tel Aviv, Israel, September 2012.

17. *Keilp, J.G., Gorlyn, M., Burke, A.K., Oquendo, M.A., Mann, J.J. Intact performance by suicide attempters on a ventral prefrontal task. Poster presented at the annual meeting of the Society of Biological Psychiatry, Philadelphia, May 2012.

18. Gorlyn, M., Keilp, J.G., Burke, A.K., Oquendo, M.A., Mann, J.J. Neurocognitive impairment in MDD is an independent component of symptom severity**.** Poster presented at the annual meeting of the Society of Biological Psychiatry, Philadelphia, May 2012.

19. Kikuchi, T., Miller, J.M., Keilp, J.G., Schneck, N., Parsey, R.V., Mann, J.J. Abnormal Neural Responses to Incongruency in Major Depressive Disorder Compared to Controls: A Stroop fMRI Study. Poster presented at the annual meeting of the Society of Biological Psychiatry, Philadelphia, May 2012.

20. *Keilp, J.G., Gorlyn, M., Burke, A.K., Oquendo, M.A., Mann, J.J. Decomposing the nature of memory impairments in depressed suicide attempters. Paper presented at the 40th annual meeting of the International Neuropsychological Society, Montreal, Canada, February 2012.

21. Gorlyn, M., Keilp, J.G., Burke, A.K., Oquendo, M.A., Mann, J.J. Decision-making and cognitive abilities: Processing speed is integral to Iowa Gambling Task performance. Poster presented at the 40th annual meeting of the International Neuropsychological Society, Montreal, Canada, February 2012.

22. Singh, N., Keilp, J.G., Corbera, K., Fallon, B.A. Subjective Cognitive Complaints in Post-Lyme Disease Syndrome: Effects of Mood Disturbance and Actual Performance Decline. Poster presented at the 40th annual meeting of the International Neuropsychological Society, Montreal, Canada, February 2012.

23. Scheiner, D.L., Keilp, J., Burke, A.K., Oquendo, M., Mann, J.J. The contribution of posttraumatic stress disorder to explicit verbal learning and memory performance in major depression. Poster presented at the 40th annual meeting of the International Neuropsychological Society, Montreal, Canada, February 2012.

24. LeBlanc, S., Keilp, J.G., Burke, A.K., Oquendo, M.A., Mann, J.J. Smoking in depression: associations with cognition and cognitive risk factors for suicide attempt. Poster presented at the 40th annual meeting of the International Neuropsychological Society, Montreal, Canada, February 2012.

25. Perry, J.S.A., Keilp, J.G., Pelton, G.H., Stern, Y., Devanand, D.P. Prediction of conversion from mild cognitive impairment to alzheimer's disease. Poster presented at the 40th annual meeting of the International Neuropsychological Society, Montreal, Canada, February 2012.

26. *Keilp, J.G., Gorlyn, M., Harkavy-Friedman, J., Oquendo, M.A., Mann, J.J. Neurocognitive dysfunction and the nature of suicidal behavior. Paper presented at the biannual meeting of the European Symposium on Suicide and Suicidal Behavior, Rome, Italy, September 2010.

27. Gorlyn, M., Keilp, J.G., Burke, A.K., Oquendo, M.A., Mann, J.J. Iowa Gambling Task performance and suicidal behavior in Major Depressive Disorder. Paper presented at the biannual meeting of the European Symposium on Suicide and Suicidal Behavior, Rome, Italy, September 2010.

28. *Keilp, J.G., Gorlyn, M., Cooper,T., Oquendo, M.A., Mann, J.J. A modified social stress task for high-risk populations. Poster presented at the annual meeting of the Society of Biological Psychiatry, New Orleans, May 2010.

29. Gorlyn, M., Keilp, J.G., Cooper, T., Oquendo, M.A., Brent, D., Mann, J.J. Stress response in the offspring of suicide attempters: Preliminary findings. Poster presented at the annual meeting of the Society of Biological Psychiatry, New Orleans, May 2010.

30. Molero, P., Keilp, J.G., Galfalvy, H.C. Burke, A.K., Harkavy-Friedman, J.M., Oquendo, M.A. Neurocognitive effects of lithium and valproate in bipolar suicide attempters. Poster presented at the annual meeting of the Society of Biological Psychiatry, New Orleans, May 2010.

31. Gorlyn, M., Keilp, J.G., Russell, M.E.,Grunebaum, M., Burke, A.K., Mann, J.J. Antidepressant medication effects on cognitive functioning and subjective mood mediate suicidality in MDD Poster presented at the 37th annual meeting of the International Neuropsychological Society, Atlanta, GA, February, 2009.

32. *Keilp, J.G., Gorlyn, M., Russell, M.E., Harkavy-Friedman, J., Oquendo, M.A., Mann, J.J. Executive dysfunction in suicidal behavior: A correlate of violence, not attempt risk? Poster presented at the 37th annual meeting of the International Neuropsychological Society, Atlanta, GA, February, 2009.

33. Fallon, B.A., Lipkin, R.B., Corbera, K., Yu, S., Nobler, M.S., Keilp, J.G., Lisanby, S.H., Van Heertum, R., Mensh, B.D., Sackeim, H.A. Regional cerebral blood flow and metabolic rate in persistent lyme encephalopathy Poster presented at the 11th annual conference on Lyme Borreleosis and Other Tick-Borne Diseases, Irvine, CA, October, 2008.

34. *Keilp, J.G., Corbera, K., Sackeim, H.A., Mann, J.J., Fallon, B.A. Differentiating the cognitive impairments of post-treatment Lyme disease and major depression. Poster presented at the 11th annual conference on Lyme Borreleosis and Other Tick-Borne Diseases, Irvine, CA, October, 2008.

35. Shikman, M., Bruder, G., Keilp, J., Farrell, W. Demographic influences on working memory tasks in early to middle adulthood. Poster presented at the 36th annual meeting of the International Neuropsychological Society, Waikoloa, HI, February, 2008.

36. Gorlyn, M., Keilp, J.G., Grunebaum, M.F., Russell, M.E., Burke, A.K., Oquendo, M.A., Mann, J.J. Working memory performance predicts short-term antidepressant treatment response in high suicide risk patients with MDD. Poster presented at the 36th annual meeting of the International Neuropsychological Society, Waikoloa, HI, February, 2008.

37. *Keilp, J.G., Gorlyn, M., Russell, M.E., Burke, A.K., Oquendo, M.A., Mann, J.J. Neuropsychological dysfunction in suicidal behavior: Executive or information processing deficit? Paper presented at the 36th annual meeting of the International Neuropsychological Society, Waikoloa, HI, February, 2008.

38. Gorlyn, M., Keilp, J.G., Grunebaum, M.F., Taylor, B.P., Oquendo, M.A., Bruder, G.E., Stewart, J.W., Mann, J.J. Neuropsychological predictors of SSRI treatment nonresponse in depression. Poster presented at the annual meeting of the Society of Biological Psychiatry, Toronto, Ontario, CAN, May, 2006.

39. Gorlyn, M., Keilp, J.G., Oquendo, M.A., Mann, J.J. Specific deficits in semantic fluency in high-lethality suicide attempters. Poster presented at the annual meeting of the Society of Biological Psychiatry, Toronto, Ontario, CAN, May, 2006.

40. Milak, M.S., Parsey, R.V., Keilp, J., Oquendo, M.A., Malone, K.M., Mann, J.J. Neuroanatomical correlates of the subjective psychopathologic components of major depressive disorder mapped by PET: A comparison with objective psychopathologic components of major depression. Poster presented at the annual meeting of the Society of Biological Psychiatry, Toronto, Ontario, CAN, May, 2006.

41. Fertuck, E.A., Marsano, S., Keilp, J.G., Stanley, B.H., Tryon, W.W., Oquendo, M.A., Mann, J.J. The impact of anxiety and borderline personality disorder on neuropsychological performance in major depression. Poster presented at the annual meeting of the Society of Biological Psychiatry, Atlanta, GA, May, 2005.

42. Gorlyn, M., Keilp, J.G., Sullivan, G.M., Oquendo, M.A, Mann, J.J. Increased alertness with low CSF 5-HIAA in major depression. Poster presented at the annual meeting of the Society of Biological Psychiatry, Atlanta, GA, May, 2005.

43. *Keilp, J.G., Malone, K.M.M., Sackeim, H.A., Mann, J.J. Antidepressant and behavioral effects of acute serotonergic enhancement. Poster presented at the annual meeting of the Society of Biological Psychiatry, Atlanta, GA, May, 2005.

44. Dwork, A.J., Ilievski, B., Mancevski, B., Kurzon, M., Serafimova, T., Trencevska, I., Rosoklija, G., Keilp, J. Histological evaluation of prefrontal white matter in schizophrenia. Poster presented at the annual meeting of the International Congress on Schizophrenia. Savannah, GA, April, 2005.

45. Mancevski, B., Rosoklija, G., Kurzon, M., Serafimova, T., Ortakov, V., Trencevska, I., Keilp, J., Dwork, A. J. Effects of historical introduction of neuroleptics on symptomatology in chronic schizophrenia inpatients. Poster presented at the annual meeting of the International Congress on Schizophrenia. Savannah, GA, April, 2005.

46. Wyatt, G., Keilp, J.G., Oquendo, M.A., Sackeim, H.A., Mann, J.J. Executive function and dysfunction in suicidal behavior. Poster presented at the 33d annual meeting of the International Neuropsychological Society, St. Louis, MO, February, 2005.

47. Gorlyn, M., Klain, M., Keilp, J.G., Oquendo, M.A., Sackeim, H.A., Mann, J.J. Slowing of information processing speed in major depression. Poster presented at the 33d annual meeting of the International Neuropsychological Society, St. Louis, MO, February, 2005.

48. Gorlyn, M., Keilp, J.G., Oquendo, M.A., Sackeim, H.A., Mann, J.J. The WAIS-III and major depression: Absence of VIQ/PIQ split. Poster presented at the 33d annual meeting of the International Neuropsychological Society, St. Louis, MO, February, 2005.

49. *Keilp, J,G., Gorlyn, M., Oquendo, M.A., Sackeim, H.A. Mann, J.J. Attention, memory and language fluency in depression and suicidal behavior. Presented at the 33d annual meeting of the International Neuropsychological Society, St. Louis, MO, February, 2005.

50. *Keilp, J.G., Oquendo, M., Mann, J.J. Time estimation in depression and suicide. Poster presented at the annual meeting of the Society for Biological Psychiatry, New York, NY, May, 2004.

51. *Keilp, J.G., Milak, M., Parsey, R., Oquendo, M., Mann, J.J. PET correlates of neuropsychological impairment in depression and suicide. Paper presented at the annual meeting of the Society for Biological Psychiatry, New York, NY, May, 2004.

52. *Keilp, J.G., Milak, M., Parsey, R., Oquendo, M.A., Mann , J.J. Distinct patterns of cerebral metabolism associated with component symptoms of depression. Paper presented at the 9th annual meeting of the Organization for Human Brain Mapping, New York, NY, June, 2003.

53. *Keilp, J.G., Corberra, K., Fallon, B.A. Intellectual and memory impairment in chronic Lyme disease. Poster presented at the 31st annual meeting of the International Neuropsychological Society, Honolulu, HI, February, 2003.

54. *Keilp, J.G., Lowe, G., Schori, E., Sackeim, H.A., Mann , J.J. Screening for attempt-related brain damage in neuropsychological studies of suicidal behavior. Poster presented at the 31st annual meeting of the International Neuropsychological Society, Honolulu, HI, February, 2003.

55. *Keilp, J.G., Sackeim, H.A., Brodsky, B.S., Oquendo, M.A., Mann , J.J. Neuropsychological correlates of suicide risk and attempt severity. Paper presented at the 31st annual meeting of the International Neuropsychological Society, Honolulu, HI, February, 2003.

56. *Keilp, J.G., Sackeim, H.A., Brodsky, B., Oquendo, M.A., Mann, J.J. Neuropsychological dysfunction in depression: Associations to suicide risk indicators and severity of suicidal behavior. Paper presented at the fifteenth annual New York State Office of Mental Health (OMH) research conference, Albany, NY, December, 2002.

57. Rodenhiser, J., Keilp, J., Abi-Dargham, A., Kegeles, L., Parsey, R., Eftychiou, N., Laruelle, M. Dopamine transmission parameters and personality traits: An in vivo imaging study. Poster presented at the annual meeting of the International Congress on Schizophrenia Research, Boulder, CO, March 2001.

58. *Keilp, J.G., Mann, J.J., Sackeim, H.A. Neuropsychological correlates of suicidal behavior. Paper presented at the thirteenth annual New York State Office of Mental Health (OMH) research conference, Albany, NY, December, 2000.

59. Abi-Dargham, A., Gil, R., Mawlawi, O., Hwang, D., Kochan, L., Lombardo, I., Rodenhiser, J., Kegeles, L., Martinez, D., Keilp, J., VanHeertum, R., Gorman, J., Laruelle, M. Selective alterations in D1 receptors in schizophrenia: A PET in vivo study. Paper presented at the annual meeting of the American College of Neuropharmacology, San Juan, Puerto Rico, December, 2000.

60. Mann, J.J., Oquendo, M.A., Parsey, R.V., Campbell, C., Keilp, J., Cooper, T.B., Malone, K.M., Kegeles, L., Slifstein, M., Van Heertum, R. PET imaging of serotonin responsivity in bipolar and unipolar mood disorders. Paper presented at the annual meeting of the Society for Nuclear Medicine, May, 2000.

61. Kegeles, L., Zea-Ponce, Y., Abi-Dargham, A., Rodenheiser, J., Wang, T., Keilp, J., Weiss, R., Cooper, T.B., VanHeertum, R., Mann, J.J., Laruelle, M. Reproducibility of striatal amphetamine-induced dopamine release with [^{123}I]IBZM SPECT in healthy subjects. Poster presented at the annual meeting of the Society for Neuroscience, Los Angeles, CA, November, 1998.

62. Fallon, B.A., Keilp, J., Prohovnik, I., Mann, J. Lyme disease vs. depression vs. somatization: Cognitive tests and functional imaging. Paper presented at the IXth Annual International Scientific Conference on Lyme Borreliosis, Boston, MA, April, 1996.

63. Rosoklija, G., Hays, A.P., Latov, N., Sadiq, S.A., Kaufman, M., Waniek, C., Keilp, J.G., Prohovnik, I., Dwork, A.J. Subicular MAP-2 immunoreactivity is diminished in a variety of psychiatric disorders. Poster presented at the annual meeting of the Society for Biological Psychiatry, New York, NY, May, 1996.

64. Dwork, A.J., Keilp, J.G., Waniek, C., Liu, D., Susser, E., Kaufman, M., Prohovnik, I. Alzheimer's-type pathology in schizophrenia with dementia. Paper presented at the annual meeting of the Society for Biological Psychiatry, New York, NY, May, 1996.

65. Prohovnik, I., Sano, M., DeVivo, D., Hurlet, A., Keilp, J.G., Piomelli, S. Frontal lobe dysfunction in sickle-cell anemia. Paper presented at the 17th International Symposium on Cerebral Blood Flow and Metabolism, Cologne, Germany, February, 1995.

66. Prohovnik, I., Keilp, J.G., Huey, E., Wu, A. The AD parietal deficit reflects deterioration more than current status. Paper presented at the 17th International Symposium on Cerebral Blood Flow and Metabolism, Cologne, Germany, February, 1995.

67. *Keilp, J.G., Prohovnik, I. Estimated IQ decline related to the severity of parietotemporal perfusion deficits in Alzheimer's disease. Paper presented at the annual meeting of the International Neuropsychological Society, Seattle, WA, February, 1995.

68. Cornblatt, B., Bergman, A., Wolf, L., Keilp, J., Osgood, G., Keefe, R., O'Brien, J. The Elmhurst First Episode Project: Overview and preliminary findings. Paper presented at the annual meeting of the Society for Biological Psychiatry, San Francisco, CA, May, 1993.

69. Wolf, L., Keilp, J., Obuchowski, M., Osgood, G., Keefe, R., O'Brien, J., Cornblatt, B. Neuropsychological differentiation among psychotic adolescents. Poster presented at the annual meeting of the Society for Biological Psychiatry, San Francisco, CA, May, 1993.

70. Hermann, C., Keilp, J., Lee, H., Quinlan, D., Valance, J., Herrera, J., Mohs, R. Memory activation in Alzheimer's Disease with ^{99m}Tc-HMPAO. Poster presented at the annual meeting of the Society for Biological Psychiatry, San Francisco, CA, May, 1993.

71. Intrator, J., Keilp, J.G., Dorfman, D., Bernstein, D., Schaeffer, C., Wakeman, J., Harpur, T., Hare, R., Handelsman, L., Stritzke, P. Patterns of cerebral activation in psychopaths processing affective and neutral words using single photon emission tomography (SPECT). Poster presented at the annual meeting of the Society for Biological Psychiatry, San Francisco, CA, May, 1993.

72. Cornblatt, B.A., Keilp, J.G. Attention deficits and disruptions of frontal-striatal brain functioning in schizophrenia. Poster presented at the annual meeting of the International Congress on Schizophrenia Research, Colorado Springs, CO, April, 1993.

73. Wolf, L.E., Keilp, J.G., Keefe, R., Osgood, G., Khanna, P., O'Brien, J.D., Cornblatt, B.A. Neurocognitive profiles of probable schizophrenia in adolescence. Poster presented at the annual meeting of the Society for Biological Psychiatry, Washington DC, May, 1992.

74. *Keilp, J.G., Sweeney, J.A., Wolf, L., Haas, G. Associations between ventricular enlargement and neuropsychological deficit in schizophrenia: Managing the confounding effects of neuroleptic medications. Poster presented at the conference "Neuropsychology at the Interface of Neurology and Psychiatry" sponsored by the New York Neuropsychology Group and New York Academy of Sciences; April 11, 1992.

75. Herrera, J., Keilp, J.G., Cornblatt, B., Lee, H., Stritzke, P., Valance, J., Duval, J., Davis, K. Hemispheric dysfunction in schizophrenia. Poster presented at the annual meeting of the Society for Biological Psychiatry, New Orleans, LA, May, 1991.

76. Sidtis, J.J., Thaler, H., Brew, B.J., Sadler, A.E., Keilp, J.G., Aronow, H.A., Price, R.W. The interval between equivocal and definite neurological signs and symptoms in the AIDS Dementia Complex (ADC). Paper presented at the V International Conference on AIDS, Montreal, Canada, June 1989.

77. Price, R.W., Brew, B.J., Sidtis, J.J., Sadler, A.E., Keilp, J.G., Wolf, W., Birnhak, L. A system for staging the AIDS Dementia Complex: Correlations with neurological and neuropsychological assessments. Poster presented at the V International Conference on AIDS, Montreal, Canada, June 1989.

78. Sidtis, J.J., Sadler, A.E., Keilp, J.G., Brew, B.J., Aronow, H.A., Price, R.W. Neuropsychological test performance in HIV-1 sero-positive patients on and off Azidothymidine (AZT). Poster presented at the V International Conference on AIDS, Montreal, Canada, June 1989.

79. Sadler, A.E., Keilp, J.G., Thaler, H., Brew, B.J., Aronow, H.A., Price, R.W., Sidtis, J.J. Test-retest performance on neuropsychological tests in a group of HIV-1 seropositive patients. Poster presented at the V International Conference on AIDS, Montreal, Canada, June 1989.

80. Sadler, A.E., Keilp, J.G., Dorfman, D., Brew, B.J., Aronow, H.A., Price, R.W., Sidtis, J.J. Neuropsychological performance as a function of AIDS Dementia Complex (ADC) severity. Poster presented at the V International Conference on AIDS, Montreal, Canada, June 1989.

81. Dorfman, D., Keilp, J.G., Sadler, A.E., Wolf, L., Price, R.W., Sidtis, J.J. Short-term memory (STM) capacity declines as a function of AIDS Dementia Complex (ADC) severity. Poster presented at the V International Conference on AIDS, Montreal, Canada, June 1989.

82. Brew, B.J., Keilp, J.G., Sadler, A.E., Krown, S.E., Anselmo, W., Birnhak, L., Sidtis, J.J., Price, R.W. Recombinant Granulocyte-Macrophage Colony-Stimulating Factor (GMCSF): Lack of neurological toxicity. Poster presented at the V International Conference on AIDS, Montreal, Canada, June 1989.

83. Aronow, H.A., Keilp, J.G., Krol, G., Brew, B.J., Sadler, A.E., Dorfman, D., Sidtis, J.J., Birnhak, L., Rottenberg, D.A., Price, R.W. Cerebral atrophy in HIV-1 infected patients: Relationship to neurological and neuropsychological measures. Poster presented at the V International Conference on AIDS, Montreal, Canada, June 1989.

84. *Keilp, J.G., Tryon W.W., Brew, B.J., Sadler, A.E., Aronow, H.A., Price, R.W., Sidtis, J.J. The Aids Dementia Complex (ADC) and impairments of ambulatory activity. Abstract presented at the V International Conference on AIDS, Montreal, Canada, June 1989.

85. *Keilp, J.G., Sadler, A.E., Wolf, L., Brew, B.J., Price, R.W., Sidtis, J.J. Impaired motor performance of HIV-1 infected patients is not due to simple fatigue. Poster presented at the V International Conference on AIDS, Montreal, Canada, June 1989.

86. *Keilp, J.G., Brew, B.J., Heyes, M., Sadler, A.E., Price, R.W., Sidtis, J.J. Tryptophan levels are unrelated to disturbances of mood in HIV-1 infected patients. Poster presented at the V International Conference on AIDS, Montreal, Canada, June 1989.

87. *Keilp, J.G., Sadler, A.E., Wolf, A., Brew, B.J., Dorfman, D., Price, R.W., Sidtis, J.J. HIV-1 patients' self-reported complaints of memory failure and their relationship to actual memory performance. Poster presented at the V International Conference on AIDS, Montreal, Canada, June 1989.

88. Haas, G.L., Sweeney, J.A., Keilp, J.G., Frances, A.J. Sex differences in neurocognition of schizophrenia. Paper presented at the annual meeting of the American Psychiatric Association, San Francisco, California, May, 1989.

89. Sadler, A.E., Brew, B.J., Keilp, J.G., Thaler, H., Price, R.W., Sidtis, J.J. Neuropsychological performance as a function of severity of cognitive, motor, and behavioral dysfunction in the AIDS dementia complex (ADC). Poster presented at the IV International Conference on AIDS, Stockholm, Sweden, June, 1988.

90. Brew. B.J., Keilp, J.G., Sweeney, J.A., Walsh, V., Price, R.W. Eye movement abnormalities in HIV infected patients: A marker of early neurologic involvement? Poster presented at the IV International Conference on AIDS, Stockholm, Sweden, June, 1988.

91. *Keilp, J.G., Sadler, A.E., Brew, B.J., Price, R.W., Sidtis, J.J. The effect of distress and depression on neuropsychological test performance in the AIDS dementia complex (ADC). Paper presented at the IV International Conference on AIDS, Stockholm, Sweden, June 1988.

92. DeMeo, M.D., McBride, P.A., Keilp, J.G., Tierney, H., Brown, R.P., Kream, J., Mann, J.J. Fenfluramine-stimulated prolactin release and age related differences in depressed inpatients vs. controls. Poster presented at the annual meetings of the Society for Biological Psychiatry and American Psychiatric Association, Chicago, Illinois, May, 1987.

93. McBride, P.A., Brown, R.P., DeMeo, M., Keilp, J.G., Stanley, M., Mann, J.J. Platelet 5-HT2 receptor measures: Major depression and suicide. Paper presented at the annual meetings of the Society for Biological Psychiatry and American Psychiatric Association, Chicago, Illinois, May, 1987.

94. Brown, R.P., Stipetic, M., Linnoila, M., Keilp, J.G., Stanley, M., Mann, J.J. CSF monoamines and depressive subtypes. Paper presented at the annual meeting of the Society for Biological Psychiatry, Chicago, Illinois, May, 1987.

95. *Keilp, J.G., Raduns, C., Evans, S., Brown, R.P., McBride, P.A., Mann, J.J. Psychopathology of parasuicidal and failed suicidal attempters. Poster presented at the annual meetings of the Society for Biological Psychiatry and American Psychiatric Association, Chicago, Illinois, May, 1987.

96. Sweeney, J.A., Keilp, J.G., Jacobsen, P., Solomon, C., Deck, M., Mann, J.J. Negative symptoms and brain damage in schizophrenia. Poster presented at the annual meeting of the American Psychiatric Association, Washington, D.C., May, 1986.

97. *Keilp, J.G., Sweeney, J.A., Jacobsen, P., Solomon, C., Deck, M., Mann, J.J. Negative symptoms, neuropsychological impairment, and brain damage in schizophrenia. Paper presented at the annual meeting of the Society for Biological Psychiatry, Washington, D.C., May, 1986.